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Spa Typing of *Staphylococcus aureus* Strains Isolated From Clinical Specimens of Patients With Nosocomial Infections in Tehran, Iran

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Abstract

Background: The incidence of nosocomial *Staphylococcus aureus* infection is increasing annually and becoming a true global challenge. The pattern of *Staphylococcus aureus* protein A (spa) types in different geographic regions is diverse.
Objectives: This study determined the prevalence of methicillin-resistant *S. aureus* and different spa types in *S. aureus* clinical isolates.
Materials and Methods: During a six-month period, 90 *S. aureus* isolates were recovered from 320 clinical specimens. The in vitro susceptibility of various *S. aureus* isolates to 16 antibiotic discs was assessed using the Kirby-Bauer disk diffusion method. Molecular typing was carried out with *S. aureus* protein A typing via polymerase chain reaction.
Results: The frequency of methicillin-resistant *S. aureus* in our study was 88.9%. Twenty-three (25.5%) isolates were positive for panton-valentine leukocidin encoding genes. *S. aureus* presented a high resistance rate to ampicillin (100%) and penicillin (100%). No resistance was observed to vancomycin, teicoplanin, or linezolid. The rates of resistance to the majority of antibiotics tested varied between 23.3% and 82.2%. The rate of multidrug resistance among these clinical isolates was 93.3%. The 90 *S. aureus* isolates were classified into five *S. aureus* protein A types: t037 (33.3%), t030 (22.2%), t790 (16.7%), t969 (11.1%), and t044 (7.7%). Eight (8.9%) isolates were not typable using the *S. aureus* protein A typing method.
Conclusions: We report a high methicillin-resistant *S. aureus* rate in our hospital. Additionally, t030 and t037 were the predominant spa-types among hospital-associated *S. aureus*. Our findings emphasize the need for continuous surveillance to prevent the dissemination of multidrug resistance among different *S. aureus* protein A types in Iran.

Keywords: Spa Typing, Nosocomial Infection *Staphylococcus aureus*, Methicillin-Resistant *Staphylococcus aureus* (MRSA)

1. Background

A leading cause of nosocomial infection, *Staphylococcus aureus* is responsible for many conditions, including wound infections, food poisoning, osteomyelitis, and endocarditis, as well as life-threatening diseases, such as pneumonia and bacteremia (1). This bacterium is characterized by its remarkable ability to acquire resistance to antimicrobial agents, especially methicillin. In particular, methicillin-resistant *S. aureus* (MRSA) has recently emerged as a major public health concern. Methicillin was the first therapeutic option developed to treat infections caused by penicillin-resistant *S. aureus* (2).

The first MRSA isolate was reported in 1961 in the United Kingdom (3, 4). Since then, studies have revealed a steady increase in the incidence of MRSA infection. Methicillin resistance reportedly arises from the expression of a methicillin-hydrolyzing β-lactamase or the expression of an altered form of penicillin-binding protein-2 (PBP2a, also referred to as PBP2′) that is mediated by the *mecA* gene. This gene is carried within a mobile genetic element known as staphylococcal cassette chromosome *mec* (*SCCmec*) (5).

MRSA infection is currently an important cause of morbidity and mortality in both community and healthcare settings due to its resistance to nearly all currently available beta-lactam antibiotics and other therapeutic options, such as macrolides, lincosides, and aminoglycoside (6, 7). The dissemination of MRSA with multi-resistance genes has significantly limited the choice of therapeutic options available to treat staphylococcal infections, which are associated with poor clinical outcomes (1, 7). Hospital-associated MRSA (HA-MRSA) strains are usually resistant to many antibiotics and may carry virulence genes, such as the *pvl* gene, which encodes panton-valentine leukocidin (pvl). pvl is a putative virulence factor that has been hypothesized to enhance the bacterium's ability to cause severe infections in human and animal hosts (8).